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The WetNet: What the Oral Polio Vaccine Hypothesis Exposes about Globalized Interspecies Fluid Bonds

Abstract: *The author analyzes the aftermath of Edward Hooper's suggestion that the trial of an oral polio vaccine (OPV) in the Belgian colonies of Africa engendered the pandemic form of the AIDS virus, HIV-1. In response to Hooper's book, The River (1999), the Royal Society in London held a conference to debate the origins of HIV. Examination of the quick dismissal of the OPV theory opens a space for legitimately challenging the widely held belief that the vaccine contamination question was convincingly resolved. This article interrogates the relationship between historiography and the making of scientific facts and history, suggesting that historians have been too credulous of scientists' testimony. The further result of the lack of a thorough analysis of the evidence backing the OPV hypothesis has resulted in a missed opportunity to read The River as one of the few detailed accounts of the immense social, political, technological, and interspecies infrastructure constituted by Cold War vaccine production. This biomedical infrastructure dramatically changed the geographic and interspecies mobility of viruses in ways that may be impossible to reconstruct. Yet these potential transmission routes remain crucial to acknowledge. The COVID-19 pandemic draws attention to the critical importance of studying The WetNet, a concept coined by the author to name the conceptual and material infrastructures of inter- and intraspecies fluid bonding. [HIV origins, The WetNet, experimentality, Africa, zoonosis]*

Introduction

The press and scientific literature consistently present the “natural transfer,” “bushmeat,” or “cut hunter” theory, based on phylogenetic computer modeling of hypothesized mutation rates of HIV, as explaining the origin of the HIV/AIDS

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epidemic. This hypothesis posits that a simian immunodeficiency virus (SIV) entered a human sometime in the early 20th century through an animal bite or bushmeat consumption. The virus spread and mutated unnoticed for decades through sexual and medical contact, eventually giving rise to the AIDS pandemic. In various elaborations, this origin narrative elegantly combines—albeit speculatively—science and social history to account for the various HIV types and sub-types (Gilbert et al. 2007; Sharp and Hahn 2011; Worobey et al. 2003, 2016).¹

In 1999, British journalist Edward Hooper described an alternative theory, also tastefully interdisciplinary, but with a twist: The species cross-over stemmed not from so-called natural events but from the specific actions of (then) still-living scientists (Hooper 2000c). Over the course of nearly 1,000 spell-binding pages, Hooper describes how an oral polio vaccine (OPV) trial undertaken by American scientists in central Africa between 1956 and 1960 (Courtois et al. 1958; Hooper 2001c; Plotkin et al. 1961) might have launched the HIV pandemic. Like the phylogenetic models, Hooper works from the first known case of HIV-1 in Kinshasa (Léopoldville) in 1959. He finds a stunning geographic correlation between the OPV trials and the earliest cases of HIV, presents a comprehensive reconstruction of the chimpanzee camp and the nearby lab in Kisangani (then, Stanleyville, the base location of the trials), and details the development of the vaccine at the Wistar Institute in Pennsylvania by Hilary Koprowski. He argues that the vaccine, which Koprowski's team orally fed to about a million inhabitants of the Belgian Congo and Ruanda-Urundi, could, in theory, have led to a viral crossover through oral cuts or abrasions. He proposes other polio vaccine trials that took place in the former French colonies of Africa as sources for the minor outbreaks of HIV/AIDS (Hooper 2000c, 827–77).

The River received laudatory reviews in major press outlets (Altman 1999; Ciments 1999; Martin 2000; Trivers 2000). However, praise came to a swift end after a Royal Society conference (September 11–12, 2000) convened to discuss the theory in London (The Royal Society 2001). The precipitous and, I suggest, premature, rejection of Hooper's hypothesis was announced to the press during the conference, leading to its near-universal dismissal and designation even as conspiracy theory (rather than, say, as a plausible counterfactual hypothesis). Hooper and his remaining supporters were quickly excluded from subsequent discussions in the scientific press.²

In what follows, I neither recite Hooper's account nor argue that the vaccine trials launched the AIDS epidemic. Rather, I analyze how the genealogy of the OPV hypothesis's dismissal demonstrates that the closure of the debate precluded discussion, fact-finding, and uptake of the key, and very much needed, contributions of Hooper's research. This elision has crucial scientific and policy implications. Certainly it matters for the history of the HIV epidemic. In addition, analysis of the debate over the hypothesis sheds light on the politics of knowledge production, for *The River* offers one of the very few expositions of the massive global infrastructure of post-WWII vaccinology, one that includes highly mobile geographies of human experimentation involving interspecies fluid exchanges on a scale nearly unimaginable to a lay reader. Mid-century vaccine research and manufacture relied on the commercial exchange and sacrifice of millions of primates and other animals, particularly monkeys from India, Africa, and the Philippines (Kalter and Heberling 1971). Vaccinology contributed to animal trade and care networks; Cold War and

colonial politics; technologies of refrigeration, preservation, and shipping; human populations for testing; global exchange networks for biomaterials and animal and human plasma, sera, and cells. These infrastructures were more and less acknowledged in the high-stakes, fragile, competitive, and collegial power struggles among scientists committed to controlling how debates were framed and what information was documented and shared. Critical analysis of this vaccine infrastructure seems to be considered highly threatening and has been successfully curtailed. The OPV case offers an example of how.

“Fluid bonding” refers to non-barrier intimate relations through which fluid sharing occurs. In polyamory relationships, the term acknowledges a broader network: One can be fluid bonded with one’s partner’s partners. This term explicitly recognizes that fluids can carry disease and that discussion and consent are advisable; it provides language to begin the conversation. It’s a useful term, I think, in considering bonds among humans and animals that can carry disease through such quotidian occurrences as sneezing and meat consumption. Similarly, and yet practically invisibly, vaccines have required animal biomaterials on which viruses have been grown and through which they have been passaged, often with little understanding by both scientists and those vaccinated of what kinds of fluid bonding relationships were being created. By opening bodies to each other, biomedical and technological infrastructures choreographed zones that fundamentally altered and expanded viral dynamics and exchanges.

The biomedical infrastructure undergirding vaccine research and production spurred justification of these risks; assurances of the positive potential and intent of biomedicine became the foil and norm against which real or possible hazards have been adjudicated, understudied, and sometimes silenced. (To note this fact is not to take an anti-vax position.) I argue that the OPV hypothesis can be understood in this context, not exclusively for its provability, but as a plausible counterfactual. As such, this article aims to open the door to further study of what I’m calling The WetNet, a conceptual space that names an infrastructure by which fluid exchanges—some purposeful others accidental, some known others unknown or unknowable, and all living in the hyphen of nature–culture—take place and are justified, explained, or ignored. In the conclusion, I further introduce the term The WetNet to name this process. COVID-19 has brought new awareness of these infrastructures, and a new urgency to research The WetNet.

To make these arguments, I consider uncertainty as a live element in projects of historical reconstruction. Catherine Gallagher (2018) defines counterfactual histories as “what if” and “but for” scenarios. Such modeling, when applied to possible vectors of disease, can identify the architecture of trust relied on in historical mapping: *If* an iatrogenic spill-over had occurred through The WetNet, how *would* anyone know? What kinds of information, not included in extant scientific reports and publications, would be necessary to reconstruct such events? A counterfactual method offers a way forward that allows for uncertainty, enabling consideration of the multiple possibilities resulting from complex biological exchanges devised by scientists in conditions that even they, in hindsight, may admit were naive. It also offers a way to understand how allied practices, such as record-keeping, regulation, and shared assumptions, impact how the origins of emerging diseases can be reconstructed. Given how little critical social science research exists on the enormously

complex and crucially important area of vaccinology, and given the burgeoning interest in medical anthropology on zoonosis (Keck and Lynteris 2018), I believe the OPV-HIV story provides insights that increase awareness of and languages for describing The WetNet as an infrastructure of viral mobility, and, more specifically, the complex global bio-formations constituted by mid-century vaccinology.

I base the following historical ethnography on the recording of the meetings archived at the Royal Society Library; interviews with two spectators (Elizabeth Tilly and Vinh-Kim Nguyen); interviews with participants Edward Hooper, Stanley Plotkin, and Robin Weiss; a comprehensive analysis of *The River* and the papers from the conference in *Philosophical Transactions* (The Royal Society 2001); a review of the scientific literature on the hypothesis published before and after the controversy's closure; and study of primary and secondary literature in vaccine history.

The Oral Polio Vaccine Hypothesis

The River parses an astonishing array of primary and secondary documents; Hooper's materials range from flight schedules to chimp behavior to dozens of interviews with scientists and others who were involved in, or linked to, the trials and the early investigations into the first cases of HIV. The hypothesis forwards two distinct components. First, he provides arguments and evidence about why routes of HIV transmission based on human mobility and medical and sexual practices proposed by other scholars' lack credibility. He also documents uncanny geographic correlations, whereby "all 46 documented instances of HIV-1 infection from Africa through 1980 come from within 140 miles of CHAT [the OPV vaccine] vaccination sites" and "70% of these earliest AIDS cases come from a town or village where CHAT had been vaccinated" (Hooper 2001a, 806). This and other evidence provide circumstantial evidence for the vaccines as a plausible source of the initial spill-over events. Second, the tissue cultures on which the polio virus was grown offer one sensical explanation of the *mechanics* of a spill-over. For instance, the seed lots of vaccine made at Wistar could have been further passaged in locally harvested and prepared chimpanzee kidney tissue cells or alternatively, the vaccine might somehow have been contaminated with fluids from chimpanzee dissections.

In the United States and Europe in the 1950s and '60s, the renal tissues of various monkey species were used for a range of medical and virological purposes, requiring the sacrifice of vast numbers of animals (Ahuja 2013; Bookchin and Schumacher 2004). Hooper interviews several experts who verify that animal kidney tissue cultures would contain lymph and other fluids that could harbor viruses. Chimpanzees and other apes generally did not contribute organs for tissue cultures in the United States, not due to any biological barrier, but rather because the animals were expensive and dangerous. However, in the Congo, chimpanzees were in plentiful supply, and Lindi Camp, near the Stanleyville lab, housed between 400 and 600 chimpanzees (1956–59), most of whom were sacrificed as part of the Wistar-run OPV trial and other local experimentation without explanation (Hooper 2000b, 2000c, 2001a, 2001b). Hooper identified these chimps, tracked where they might have been captured, and interviewed African lab technicians who claimed that they had been making OPV with chimpanzee tissues. Additionally, Hooper identified a Polish

veterinarian, Alexandre Jezierski, who was at the time making tissue cultures from various primate tissues (including chimpanzee) at a veterinary lab nearby, and with whom Koprowski had met during a visit to the Congo.³

If viruses harbored in chimps had contaminated the vaccine and instigated species cross-over events, circumstances would have militated against any such medical recognition. For one thing, as Koprowski himself readily admitted, follow-up with trial participants was lax by design—even though Koprowski had selected rural, medically under-served areas for testing a vaccine containing strains of live polio virus whose well-known danger is the risk of spreading polio. Congo's unexpected independence in 1960 resulted in the departure of most Westerners—although to be sure the United States maintained covert military operations in the country (perhaps including the Stanleyville lab) for strategic mining and Cold War reasons (van Reybrouk 2014).⁴

Suffice it to say that researchers at the time would not have linked the polio vaccine to early AIDS (a then-unidentified illness) cases if—as would have been the case in Hooper's scenario—the few illnesses occurred would have presented as more readily diagnosed pneumonia or TB. If the virus had to be transmitted one or more times to become virulent, virtually no clinician or researcher would have made the link. The discipline-wide, broad-based intellectual framework necessary to have recognized the possibility that a virus could contaminate tissue cultures and then be spread through vaccines and gain virulence after spreading almost certainly would not have existed—even if the trial had taken place in the United States and under tight regulations. The case of the monkey virus SV40 having contaminated vaccines, as I explain below, demonstrates the resistance in the scientific community to acknowledging the dangers of animal viruses in tissue cultures. And as examples such as the synthetic estrogen diethylstilbestrol (DES) and thalidomide have shown, scientific methods and interests still tend not to orient toward understanding multi-generational effects of medical and industrial interventions.

In *The River*, Hooper relays conversations with sympathetic interviewees. However, he gained only one strong scientific-insider ally during the course of his research. Bill Hamilton, a well-respected professor of evolutionary biology at Oxford University became a proponent of the OPV theory and it was he who proposed the Royal Society conference. He never made it to the event that he initiated: He died in March 2000 from an illness contracted in the Congo while conducting research on the OPV question. His death undoubtedly had ramifications for the direction that the Royal Society conference took, as it left Hooper with no one solidly inside the establishment with an investment in the theory. While this point speaks to science and technology studies (STS) debates about controversy resolution, the existential overtone hints at the potentially significant ramifications of coincidental events in the course of history.⁴

The complexity, detail, and novelty of Hooper's theory cannot be overstated. While the length of the book may deter some readers, it would have had to be hundreds of pages longer than any of the scientific reports related to the oral polio trials for it to effectively track and explain to a non-specialist audience the history of the vaccine and the various ways in which the trial, the virus, and the cross-species contamination might have played out. Indeed, as I argue below, the controversy highlights how conflicting demands and requirements about evidence and burdens

of proof measure against assumptions about normative and reasonable behaviors and expectations in the construction of historical truths.

The Conference

The Royal Society meeting participants divide into three main groups: (1) four of the scientists associated with the Congo Trials (Paul Osterreith, Jan Desmyter, Hilary Koprowski, and Stanley Plotkin) and allies including a group of phylogeneticists; (2) Hooper and allies; and (3) a varied group of speakers addressing zoonosis generally and epidemics broadly related to HIV. The latter group augmented the “conference” milieu but didn’t contribute to the ostensible debate at hand.

The agenda was skewed from the get-go. No one but Hooper could bolster the OPV hypothesis with additional facts or evidence, and he had the same time allotment as any other speaker. His main allies consisted of the Australian sociologist of science Brian Martin who gave a paper on the notion of proof in science (Martin 2001a), and Walter Nelson-Rees (2001), a well-known scientist active in publicizing cell-line contamination, who gave rather damning testimony on the believability of the Wistar scientists.⁵

Hooper’s paper (2001a), dense with detail, among other things tracks the numbers of chimpanzees at different research sites, documents interviews with the scientists and lab technicians working in Central Africa in the 1950s, and offers evidence suggesting both that chimpanzee kidneys were being extracted and sent to the Wistar Institute and that batches of the polio vaccine were being made in Africa.⁶ His paper addresses further issues related to chimpanzee subspecies and the geography and timelines of the OPV theory versus phylogenetic modeling, and it defends against other possible arguments against the theory.

Stanley Plotkin, who would become a giant of 20th-century vaccinology, had in the 1950s just launched his career at Wistar as a junior researcher and had traveled to the Congo to help administer the trial. His paper refutes the OPV theory not with independent records of how the vaccine was made, but with the flat denial that any chimp tissues were sent to Wistar. He writes: “I was in the laboratory [at Wistar] from August 1957 to June 1961, and never saw or heard of chimpanzee cells” (Plotkin 2001, 816). He concludes, “*The River* has been praised for its precise detail and wealth of footnotes, but one can be precise without being accurate” (Plotkin 2001, 822). On the other hand, Belgian scientist Paul Osterrieth worked at the lab in Stanleyville where the Wistar scientists did vaccine efficacy and other testing on chimpanzees. He claims in his paper that: “It is true that six minced chimpanzee kidneys were sent to the Wistar Institute” (Osterrieth 2001, 839). Such discrepancies in personal recollections stand *in lieu* of records, with no wider or structured attempt at rebuttal or reconciliation. As a result, the reader has no way to judge the veracity or likelihood of the different narratives.⁷

One of the most crucial points made at the conference all but sneaked out of the building through a fire escape; certainly, it was not reported in the press. At the meeting’s conclusion, long after the reporters had left, the chair and convener of the meeting, Robin Weiss, an expert in retroviruses and cross-species viral transmission, stated that experimental vaccines *could* credibly have been the cause of the zoonosis that resulted in HIV. He wrote:

To reduce the argument over the origins of HIV to the OPV hypothesis versus the cut hunter hypothesis is an over simplistic and false antithesis. Both natural and iatrogenic transmission of many retroviruses, including HIV, have been thoroughly documented and are not mutually exclusive. Exactly how, when, and where the first human(s) became infected with the progenitor of HIV-1 group M, which gave rise to the pandemic strain, is likely, however, to remain a matter of conjecture. (Weiss 2001a, 947)

Surely this is worth truly understanding, we can hear Weiss intimating. However, as I conclude in studying the conference, closure on the OPV–HIV debate was achieved not based on the evidence (which was inconclusive) but because of the insistence of the scientists involved in the trials.

And yet, had the goal of the Royal Society meeting been to truly consider the OPV hypothesis, it is notable that many of Hooper's key points were not taken up or addressed at all by the speakers and the resulting collection of essays. No other formal structures for investigation—such as through law or a third party—were or are available to address questions of this kind or scale, and no independent researchers emerged to take on the considerable effort and potential professional risk of continuing, or verifying, Hooper's research, which included a list of follow-up research proposals such as sequencing blood samples of then still-living HIV-positive recipients of the CHAT vaccine.

Even a cursory reading of the Royal Society's conclusions (i.e., the document produced by conference convener Robin Weiss) renders problematic a ready acceptance of the conclusion that the OPV theory had been debunked at the conference. Weiss's paper can be read as a clear warning about the possibilities of zoonosis, and reading-between-the-lines, his prevarications relay an ambivalent finale. Indeed, Weiss explicitly echoes journalist Tom Curtis, who had originally introduced the OPV hypothesis in a 1992 article: "If the Congo vaccine turns out not to be the way AIDS got started in people, it will be because medicine was lucky, not because it was infallible" (Curtis 1992, 108). It's telling that while Koprowski sued Curtis for libel, Weiss's finding flew under the radar. I suggest below that this is largely because of his rhetorical style and approach.

One final epitaph to the OPV hypothesis bears noting. Hoping to confirm his hypothesis, Hooper had advocated for any extant vaccine to be tested by a neutral third party. After the conference, samples provided by Wistar tested negative for chimp DNA and SIV/HIV virus. The Wistar scientists claimed absolution, the press once again declared the case closed, and Weiss "jumped the fence on the polio vaccine hypothesis in favour of 'disproved.'"⁸ For his part, Hooper pointed out flaws in the testing, most specifically that, "There is no evidence that any of the CHAT samples produced at the Wistar Institute and Wyeth Laboratories, . . . have any relevance to the vaccinations conducted in Africa." He adds: "it is now apparent that the vaccine used in Ruzizi and along Lake Tanganyika did not comprise one homogeneous preparation of CHAT pool 10A-11 [the pool that was tested], but rather several different CHAT preparations, made at different times and originating from different laboratories" (Hooper 2000a, 2001a, 807). None of the CHAT samples tested had been used in Africa. While even Koprowski had claimed that

samples of the vaccine no longer existed (Vaughan 2000), this testing pounded the final nail into the coffin.

If consensus science worked because scientists have a great deal of cultural and economic capital that they used to guide the debate, and journalists and historians generally fell in line, it remains true that free and open debate of the OPV theory would have required institutions, medical record-keeping practices, truly independent peer review, and modes of interrogation that simply did not exist then and still may not exist. For all the many reasons to critique legal reasoning and practice, the legal system offers a version of a structure with the express purpose of determining historical likelihoods based on weighing various forms of evidence and testimony. Clearly, if capital-S science, or capital-M medicine *had* wanted to develop a means of self-regulation, ample opportunities and models have been presented over the decades. Arguably, Hooper's hypothesis *could* have been scrutinized in accordance with scientific methods, which would have required a serious investigation of his points. In any case, whether because there was no formalized way to handle a narrative such as Hooper's, or because there was no way to force the OPV hypothesis into the self-regulatory pathways of a scientific method (i.e., how to gain funding for such an undertaking?), personal responses and judgments took on an outsized role, and slippages and contradictions in the record remain uncontested.

The Final Report: Ambivalent Intentions

Academic conferences typically gather independent researchers to present work on overlapping interests, and, as such, are not intended to resolve controversies in any structured or rigorous way. Thus, a conference offers a curious format to tackle a subject of such complexity, and Robin Weiss's published paper assessing the proceedings similarly offers a problematic summation, one that neither provides the evidence nor the logic to adequately conclude the debate despite its presentation as such. In his essay and in person, Weiss represents the two-day Royal Society meeting as an open and rigorous debate whose aim was to "lay open all the arguments and counter arguments."⁹ One can only guess at the reasons for this rush to closure in a mere two days. Weiss had already reviewed *The River for Science*, where he described it as "a towering achievement; right or wrong in its main conclusion, there is much to learn from Hooper's exposition" (Weiss 1999). As such, Weiss also mentions a second tier of "important lessons to be learned from Hooper's analysis," which he lists as "our complacency over 44 years' use of primary monkey kidney cells as a substrate for live viral vaccines" and the use of litigation to shut-down debate, as Koprowski had done by suing Tom Curtis and had threatened to do to Hooper.

Weiss's conclusion to the proceedings employs an intriguing method to leave the door ajar for future consideration of the OPV theory while at the same time appearing to reject it outright (Weiss 2001a). After each point Weiss makes in favor of the cut hunter theory, he curiously loops back to note that none of his points disproves the OPV hypothesis. Such rhetorical skill, I would argue, was a crucial factor in the closure of the debate over OPV as a source of HIV. It also suggests that subsequent commentators did not closely read the document and as such, the debate was closed largely on the basis of the press reports. Weiss's argument consists of a

series of subjective assessments: his trust in the scientists' testimony; his view that the OPV theory seems "contrived"; and his belief that the burden of proof lies with Hooper.

Weiss finds no motive or evidence for a cover-up on the part of the scientists: He finds them believable, falling squarely into a kind of old-school notion of reasonableness as described by Shapin and Schaffer in their classic work on experimental science (Shapin and Schaffer 2011). Weiss also discusses what he considers to be the unassailable reputation of the pharmaceutical industry. Ultimately, in themselves both these points are irrelevant to the historical possibility of the OPV theory. But crafting objectivity was an accomplishment of personality and rhetoric, and, as a result, the entire edifice of the conference depended on the believability and characterization of Koprowski as a disinterested bystander, genuinely wanting to engage a debate over whether or not he caused an HIV pandemic that has killed tens of millions of people through his poorly run trial (with no control group and no plan for follow-up) on medically underserved colonial people.

Weiss clarified to me in an interview his reasons for trusting the Wistar scientists' word, despite the extremity of the claim against them. *In the mid-1950s*, he explained, it would have been completely acceptable for the scientists to have used chimpanzee tissues for vaccine manufacture.¹⁰ This reasoning (they are honest because it was acceptable practice to do the very thing that is purported to be a root cause in the OPV theory), enables him to both embrace the possibility of OPV transmission and retain the credibility of the scientists involved in these trials. Weiss offered another confusing premise. He writes, "Neither does the polio vaccine industry have a particularly bad record of cover-up" (Weiss 2001a, 952). Leave aside that no unitary polio vaccine industry existed at the time, what industry there was had virtually nothing to do with Koprowski's trials. Still, Weiss gives two questionable examples of the "success" of the industry. He cites the Cutter Incident, in which an improperly made vaccine infected some 40,000 people with polio, resulting in 10 deaths and 200 cases of paralysis and that was aggressively defended by Cutter Labs in subsequent personal injury legal cases (Offit 2005).¹¹ Then he mentions SV40, a monkey virus that contaminated Salk's polio vaccine and was spread to millions of Americans. Beneath the public relations skein of these anecdotes, they do little to support Weiss's belief in the benevolence of the mid-century vaccine industry.

I outline the SV40 debacle below, but here it bears noting that the SV40 incident *was* more or less covered up. In other words, Cutter Lab's live polio and the SV40 contaminations both resulted from manufacturing errors and poor regulation, precisely the stakes in the OPV question. Nevertheless, Weiss offers a confounding case for how the SV40 incident should further confirm our rejection of the OPV theory. He praises the "quick response" to SV40 by describing the replacement of kidney cell substrates derived from rhesus macaques with that of African Greens in polio vaccine manufacture. Then, on the other hand, he writes that, "One could regard that as leaping out of the frying pan into the fire had SIVagm [shorthand for an SIV carried by an African Green monkey] been the source of HIV."¹⁴ He further concludes that "one cannot wholly preclude [SIV] slipping through on rare occasions considering the billions of doses administered during the last 40 years" (Weiss 2001a, 952). As he well knows, the consequences of even one rare slip could be an epidemic disease. The take-away from Weiss's points is *not* that there were

no cover-ups, but that the whole infrastructure of vaccine development, testing, and administration was highly experimental in 1950s and early 1960s, with an adventitious simian virus SV40 being widely spread, lax manufacturing protocols, unethical experimentation, little regulatory oversight, and ultimately the likelihood of SIV's "on rare occasions" slipping into vaccines.

Questions relating to subtypes and recombination lie beyond this essay's purview. Suffice it to say that amid complexity and speculation, Weiss turned to Occam's Razor, a problem-solving principle asserting that the simplest explanation is generally the correct one. Weiss argues that the OPV theory is "unnecessarily complicated" (Weiss 2001a, 949). Specifically, the diversification date of the virus according to phylogeneticists would have been the date that it entered the human species, whereas for Hooper, a number of similar viruses from chimp tissue cultures would have transferred to humans in the course of the vaccine trials of the 1956–60 period. Turning to medieval philosophy to adjudicate an issue of this magnitude offers an intriguing move. Surely, the "simplest" explanation depends on one's basic disposition or knowledge base. For some people, for example, consideration of the colonial and neo-colonial relations that structured this trial, and arguably, the Royal Society's consideration of the OPV hypothesis and perhaps even the development of modern virology itself, would be essential. Here again Weiss prevaricates and allows the possibility of multiple routes of cross-species transmission; both the natural transfer and the OPV theories of the cross-over could simultaneously be true.

Ultimately, Weiss's essay, either brilliantly or disappointingly, implies that the conference has adjudicated and dismissed the OPV theory. Only by engaging the text does one see how little evidence this conclusion rests on. Barely discussing Hooper's findings, he relies instead on a strong belief in the good of science and its spokespeople. The writing may well be in bad faith, as Martin's (2010) broader reading of the conference suggests. Hedging also offers an effective form of manipulation. Weiss might have been eager to close the debate for reasons superseding the implications of the debate: fears of an anti-vax movement; the challenge of an accomplished journalist–historian outsider who was unpopular with Weiss's powerful, senior scientific colleagues; and the consequences of acknowledging the sheer magnitude of the issue. Regardless, the document surely evinces a missed opportunity to do exactly what Weiss seems to want to do (i.e., open debate on the risky practices of the era).

In the aftermath of the conference, scholars have reinforced the idea of the debate's closure. For example, in *The Origin of AIDS* (Pepin 2011), a book that has emerged as the model for the explanation for AIDS, physician and historian Jacques Pepin devotes three pages to *The River*. Pepin bases his dismissal of Hooper on Plotkin's argument and (mistakenly) accuses Hooper of a rookie mistake in confusing local dilution of concentrated vaccine stock with local production or amplification (Gellin et al. 2001; Pepin 2011, 52). Somewhat bizarrely, Pepin bases his account solely on the word of the scientists who ran the trial and accuses anyone who would doubt his reliance on the defendant scientist's account of conspiracy thinking. Specifically, in considering the Wistar vaccine sample that tested negative for chimpanzee DNA (see above), he basically accuses any reader who may question the objectivity of the Wistar scientists of being a "conspiracy theorist" (Pepin 2011,

52). Rather than engaging Hooper's actual hypothesis, he offers an *ad hominem* attack against anyone who doubts the honesty of the lab.

Here again, I'm not claiming anything about the vaccine and its link with HIV. But it's notable that a book that serves as the go-to resource for the origins of the epidemic offers so little actual evidence to substantiate its claims, and that this has consistently flown under the radar of readers and reviewers. A peculiarly titled review of Pepin's book by physician and science historian Howard Markel (2011), "*It's the Science, Stupid*," illustrates this point. Markel briefly parodies Hooper's book as "insisting" on a "fanciful thesis." He then poses Pepin's breakthrough, based on "meticulous scientific analysis," that "a viral strain called SIVcpz, which infects large numbers of . . . chimpanzees living in central Africa, was the central source of HIV-1." Hardly a breakthrough, both Hooper and Pepin agree on this point.¹² Where they differ is in their hypotheses of *how* the species jump took place. But despite Markel's assertions to the contrary, no solid evidence marks Pepin's account as specifically more "convincing" or "brilliant" than Hooper's; Markel's convictions ring hollow.

What Can't Be Asked

Why wade into this territory? Why pursue the deeper story-behind-the-story of the OPV hypothesis in relation to it being both a legitimate possibility for zoonotic events and a fascinating story of the complex intertwining of human-animal and viral interspecies transmissions quite aside from HIV?

The late scholar of historiography Hayden White finds a scholarly discipline constituted by what it *forbids* its practitioners from doing. He writes that "that the so-called 'historical method'" consists of little more than the injunction to "get the story straight" (without any notion of what the relation of "story" to "fact" might be) and to avoid both conceptual overdetermination and imaginative excess at any price (1978, 126). This useful insight helps make sense of the OPV debate, since the scientific consensus conclusion about it consists not of provable facts, but rather, as I have outlined, a historical narrative based on what commentators assume as plausible, sensible events. Pepin, Markel, and others (Cohen 2000; Natrass 2012) who dismiss the OPV hypothesis out-of-hand indicate precisely what is "forbidden" in historical scholarship about science: Historians have had difficulty disagreeing with history *as constituted by scientists*. It doesn't help that Hooper's account is organized not as a lucid explication of his results, but as a narrative of a decade-long pursuit of interviews, discoveries, and hypotheses; few readers would put in the time it takes to get through *The River*. But the same could be said for numerous historical and academic texts and archives that historians do manage to closely study and interpret. At stake is precisely the question of how scientific knowledge is formed by means of dismissing certain facts made visible by other disciplines of experts, such as, potentially, historians.

Shifting the focus slightly from an attempt to resolve what *did* happen toward the material conditions of vaccine production and what *could* have happened hints at the high cost of this hesitation. This history of vaccine infrastructure has eluded intensive study in the history of science, STS, and medical anthropology. And yet the biomedical infrastructure that enabled new routes of zoonotic and intra-species

viral transmissions has unquestionably led to new conditions of life and death for animals and humans, in relation to the viruses that flow among us. The post-WWII rise in the global animal trade required for vaccine and other biomedical products; the pooling of animal and human tissues such that one carrier of a virus could infect large quantities of product; and the cavalier attitude of scientists, all created infrastructures for viral transmissions much in need of study in themselves as well as in the context of how the dangers have been justified and apportioned. As Ed Cohen has written in thinking about the conditions leading to swine flu: “Feedlots do not constitute the only way to raise pigs, though they do provide excellent ways to breed new bacteria and viruses. . . . Moreover, why was the pig domesticated, and how does this porcine history contribute to its evolution as a ‘mixing vessel’?” (Cohen 2011, 29). Histories of agricultural production have been widely considered for how they create new possibilities for disease, whereas vaccine production notably has not.

Vaccine production in the 1950s and ’60s, did result in at least one major iatrogenic zoonotic transfer of a monkey virus, SV40, spread through Jonas Salk’s killed polio vaccine. Revisiting that story in the context of how the OPV hypothesis was handled opens some larger questions about the history of vaccinology infrastructures.

Salk completed his 1954 field trial on 1.8 million American children of a vaccine consisting of killed polio virus. Vaccine production involved growing polio virus on the kidney tissues of rhesus macaques imported from India and killing it with formaldehyde. At the time, scientists had mistakenly assumed first, that any extant monkey viruses would also be killed; second, that monkey viruses would not cross the species barrier; and/or third, that adventitious viruses weren’t important to consider (Bookchin and Schumacher 2004, 79).

A complicated and relevant story ensued. The short version is this: Bernice Eddy, a scientist working at the Laboratory of Biologics Control (LBC) since 1936 had completed award-winning work devising potency and safety tests for gamma globulin and developing influenza and polio tissue cultures. With Sarah Stewart, an NIH scientist, Eddy received international recognition and founded the field of viral oncology with her co-discovery of the SE-polyoma virus (Eddy and Stewart 1959). Having shown that a mouse virus could cause cancer in small mammals, she began to wonder if a monkey virus could cause cancer in other primates, including humans. While the occasional virologist had raised misgivings about the possibility of vaccines as a vector of zoonosis (Hull et al. 1958), no one had raised the chance that simian viruses could cause cancer. Not finding anyone at the LBC willing to collaborate on what was considered politically sensitive and possibly career-hijacking work, Eddy herself soon found that 109 out of 154 hamsters injected with rhesus kidney cell extract developed tumors and eventually died. She suspected that the tumor-causing substance was hardy and virulent, had a long latency period, and maintained oncogenicity over time and through passage from animal to animal. And it clearly originated in the monkey tissues.

At this point, Eddy presented her results to her boss, the head of vaccine safety testing at the Division of Biologics Standards, Joe Smadel. Smadel discouraged Eddy’s work, eventually forbidding her to publish without his permission (which he rarely gave), moved her into a tiny lab, and divested her of her vaccine

responsibilities. While Eddy did ultimately publish her work, Debbie Bookchin and Jim Schumacher (2004), in their detailed history of SV40, *The Virus and the Vaccine*, explicitly considered Smadel's response a cover-up.

Smadel eventually admitted the veracity of Eddy's discovery of SV40 when Ben Sweet and Maurice Hilleman (Hilleman 1998) disclosed their simultaneous finding of the same agent contaminating rhesus and cynomolgus monkey tissues.¹³ A morass of interests confounded the debates that followed among virologists over what to do about SV40: The USSR was winning the "polio gap" with a more effective, cheaper, and painless oral polio vaccine developed by Albert Sabin (Bookchin and Schumacher 2004, 70); the competition between killed and live polio vaccines backed by large personalities; manufacturers' questions regarding liability for the contamination; disparate levels of concern about SV40's potential dangers; and fear about losing public trust in a vaccine already widely distributed and celebrated.¹⁴

Keep in mind that Hilary Koprowski (the lead scientist of the polio vaccine trials in the Congo) thought it best not to exaggerate the importance of viral contamination: "If an adequate number of persons exposed to these agents have been shown to develop specific antibodies without any clinical disease, the evidence should be regarded as overwhelmingly in favor of the harmlessness of these agents" (Koprowski 1960, 975). This account clearly does not consider the chance of possible virus mutations. Once Koprowski's lab developed a human diploid vaccine strain made of fetal tissue, his opinion changed and he advocated against the use of monkey tissues (Wadman 2017); this later advocacy, rather than the former lack of concern about zoonosis, was foregrounded in the Royal Society meeting.

My point is that the SV40 scare could have led to a reconsideration of the fundamentals of the vaccine program along several lines of inquiry: the conditions of monkey importation, gang caging, sacrifice, and sterilization; the pooling of tissues; the testing of tissue cultures for contaminants. However, it did not. While vaccine companies were allowed to use up their stocks of SV40 contaminated vaccine, no plan was made for long-term testing of the 10–30 million Americans who now carried SV40, and the press at that time did not cover this hidden story about the virus. The scientific literature since then has generally accepted that SV40 was benign to humans, or at least that it had no immediate and noticeable effects. Nevertheless, among those who have carefully tracked the studies on SV40's potential impact on humans, some still find that the research done was "not sufficient to completely exclude the induction of neoplasms, chronic neurological disease, or other complications at a low frequency and after a long interval" (Lewis 1973; Shah and Nathanson 1976, 9). In other words, there remains room for doubt about the risks of zoonosis in the history of pharmaceutical production more generally.

New Viral Infrastructures

Several points make the SV40 case relevant to the analysis of the OPV theory and to my broader advocacy for research on The WetNet. One relates to the sociality of history. Namely, Eddy's discovery was ignored, and she was forbidden from reporting it at professional conferences. It is impossible to disentangle the misogyny in the field from the unwelcome content of her news. Similar omissions might have been at work in the dismissal of Hooper's findings on grounds that it was not being

done by a laboratory scientist. (Hooper was outside both institutional science and academia). It is notable that Maurice Hilleman, a white male, took on the cause of SV40 and insisted that the adventitious virus be removed from the vaccine. One can, therefore, imagine a world in which SV40 had not been identified or removed simply because it did not have the right kind of advocate, in the same way we can imagine was the case for HIV.

Similarly, one can imagine a situation in which SV40 was, or became, much more virulent. The contemporary assumptions about the sterility of animal tissues and the practices of gang caging animals and pooling of tissues from multiple animals now seem frankly naive. At the time, the dangers of pooling biological tissues were known—if not exactly addressed—given events such as the spread of hepatitis-B in the human blood and plasma supply (Allen 1969). To deal with the SV40 contamination, a decision was made to switch to African Greens (imported from Uganda). This did not solve the fundamental concern over species cross-overs, as Robin Weiss, cited above, pointed out.¹⁵ Third, no plans were undertaken for recording possible long-term or multi-generation effects of SV40.

It was not that scientists at the Royal Society meeting were unaware of this history. Walter Nelson-Rees acknowledged some of the background to the more problematic aspects of The WetNet by tracking a history of the contamination of tissue cultures that threatened to discredit much of the biological research done in the 1950s and 1960s. His paper did not directly support the OPV theory but rather illustrated some of the conditions in which biomedical research was taking place—and opened space for questioning its trustworthiness. He points to the whitewashing of tissue contamination by scientists and scientific journals alike:

To this day, cross-contamination of cultures has plagued many researchers, often leading to mistaken results, retractions of results, cover-ups and some out-and-out falsification of data and results following inadvertent use of the wrong cells. Also, during years of examining cultures for purity we learned that many virologists were not too concerned about the specificity of the cultures they used to propagate the particular virus under study as long as the substrate (whatever it might have been) gave optimal virus yield. (Nelson-Rees 2001, 849)

Nelson-Rees details numerous cases in which the cell substrates differed from what was named in reports, along with a lack of care in specifying, recalling, or recording which cultures were actually used.¹⁶

How are we to make sense of the history of these trials and the closure of the debate and concede not only the risks taken by the scientists but the possibility that they (and by extension, other scientists) caused grave, if unintended consequences in unknowable ways? The hundreds of trials involving exchanges of animal and human tissues and fluids, any one of which could in theory have spread unknown viruses resulting in epidemics, makes the need for inquiries like Hooper's all the more evident, even as the fear of anti-vaxxers make them all-the-less fundable and the unfounded derision Hooper has suffered makes them all the more unlikely.

As a study of what kinds of information would have been needed to be tracked to properly answer the questions he poses, Hooper's work draws attention to areas of silence around what science has either not been done (Clarke et al. 2016; Hess 2016) or has been inadequately documented, and how details can be misrepresented or misunderstood because of the scale and geographies of experimentation and the limited venues available for interdisciplinary debate. Understanding the OPV hypothesis as a plausible counterfactual narrative enables medical anthropologists to engage with, rather than to disavow, uncertainty. His work offers another way forward, keeping multiple narratives and possibilities alive and changing the kinds of questions that can be asked. (After all, many similar and unanswerable questions could be lobbed toward the natural transfer theory.)

Conclusion: The WetNet

I've tracked the rise and fall of an alternative history of the HIV-1 pandemic and suggested that the discrediting and subsequent lumping of the OPV hypothesis into conspiracy too easily waives a plausible origin story, one of unfathomable scale. Likely, strong personalities and back-room chitchat impacted the outcome, in addition to structural features militating against full consideration of complex data. The deliberation that took place at the Royal Society meeting tended to privilege the short, written reports and recollections of the scientists who ran the trial over the comprehensive investigation and interdisciplinary reconstruction posed by Hooper. A closing of the ranks after the meeting resulted in a situation whereby a small group of scientists controlled how, when, and what information was relayed to a broader public, with supporters of the OPV theory blocked from publishing in the scientific press. Mainstream journalists and other commentators tended to regurgitate these conclusions whole cloth.

The Wistar scientists strategically allied themselves with the endeavors to eradicate polio. Questioning the trial, their logic went, undermined the entire vaccine project. Ironically, Koprowski is widely considered the loser of the polio vaccine race; his polio vaccine was sparsely used and never licensed in the United States. The success of the scientists' multiple defenses despite their open-endedness, and the repetition of those defenses in historical and social science literature, points not only to what cannot be known about crucial aspects of the history of science experimentation because of the dearth of records, but apparently also to what could not be asked about the trials, let alone exposed—at least at the time of the Royal Society meeting.

The structure of the OPV debate suggests that precisely what cannot be interrogated is the details of an infrastructure that produced extravagant new conditions for global and interspecies viral exchanges. This infrastructure has been integral to the development and mass manufacture of vaccines. Infrastructure can be described as, "physical networks through which goods, ideas, waste, power, people, and finance are trafficked" (Larkin 2013, 327). As bits of information in search of hosts, viruses float in the interstices among bodies and can be transferred through food, door handles, and all manner of fluids. Infrastructures that bolster community, agriculture, markets, transportation, and the like serve also to "traffic" viruses creating fluid bonds among the multiple participants. I bring attention to the invisible side-effects

of this edifice—as constituting a complex infrastructure for viral transmission—by naming it The WetNet.

Vaccine research and vaccine mass production, as a key aspect of The WetNet, continue to evade critical attention, which is curious given that vaccinology’s central methods involve fortifying and attenuating viruses by moving them through animal and human tissues. This area has been difficult to access for social scientists for reasons traced here. But COVID-19 shows in spades the need for new approaches to recognizing and managing the routes of viral transmission.

It matters to how we understand the history of science in the context of colonialism and other power differentials whether the HIV-1 pandemic originated by “natural transfer” (i.e., with a person snacking on a chimpanzee elbow) or if it was spread through a vaccine made of virus grown on a slurry of primate tissues. It seems crucial to building adequate research and regulatory infrastructures in the future to know that methods exist for gathering the necessary evidence in reconstructing the history. But vaccine history is too often seen outside of its material infrastructure, as if vaccines were made of hope and genius rather than animals and viruses. To note that fact is not to be anti-vax.

Infrastructure requires both physical and rhetorical maintenance. The physical requirements for vaccine production included the availability of human test populations; tissues from hundreds of thousands of primates gleaned through the capture, transport, and sacrifice of the animals; circuits of labor from poachers to technicians, from keepers to veterinarians; government and private labs and funding; and too much else to list, all of which it helped create and on which it relied. All of this needs richer elucidation and analyses. As I tracked here, the recognition of SV40 in the 1960s could, in theory, have resulted in a revision of vaccinology’s basic infrastructures, had it been understood as a failure. Computer scientist Terry Winograd wrote of system failure as an opportunity: “A breakdown is not a negative situation to be avoided, but a situation of non-obviousness, in which some aspect of the network of tools that we are engaged in using is brought forth into visibility . . .” (Winograd 1992, 164). Certainly, as a potential (but-for) pandemic, SV40 offered an opportunity to reconsider the risks and dangers of primate tissue use. Arguably, Robin Weiss attempted to acknowledge this failure in his conclusion to the conference, while at the same time suturing the ultimate rectitude of the vaccinologists.

Bringing attention to The WetNet as an infrastructure for viral distribution enables a revision of the war metaphors ubiquitous in the COVID-19 pandemic and an attention to the networks through which human and animal lives and viruses intercalate. It offers a way to expand explanations of zoonotic events that have tended to focus on livestock farming, unsterile needles, and sexual and culinary habits. Even as these are necessary (tendencies toward xenophobia aside), they also have served as an excellent foil to reflect attention away from histories of biomedicine, technology, and unregulated experimentation.

Ultimately, the misreading of the Royal Society meeting and subsequent events as fully resolved has resulted in a missed opportunity to understand the wider contribution of *The River* as a detailed account of immense social, political, technological, and interspecies infrastructure that served as the conditions of possibility of the vaccine project writ large. Even if one finds his suggestions distasteful, Hooper offers a starting point from which to trace the complex fragility and the enormous risks

that were undertaken in 20th-century vaccinology. This is the moment for an open and honest reappraisal of the implications of *The WetNet* in its many iterations.

Notes

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1. The explanation relies on an enormous coincidence—that crossovers happened around the same time of at least five main varieties of HIV, with at least two types of primates, in different areas of the continent, and lay dormant or unnoticed for decades despite thousands of years of butchering and eating primate meat.

2. Hooper continues to publish on a website, www.aidsorigins.com.

3. Hooper has also noted that he has a taped interview with Pierre Doupagne, chief technician at the *Laboratoire Medical de Stanleyville* between 1949 and 1960 who specifically said that he had prepared tissue cultures from chimps and given them to Paul Osterrieth (Hooper 2007).

4. Koprowski lived to be 96.

5. Others sympathetic to Hooper’s case included Tom Burr, Gerry Myers, Pascal Gagneux, Julian Cribb, and Daniel Vangroenweghe. Hooper believes that Nelson-Rees, Burr, and Gagneux were pressured “not to identify too closely with the OPV hypothesis” by the conference organizers Simon Wain-Hobson and Robin Weiss (personal communication, April 13, 2020).

6. Hooper discusses his method of triangulating information sources. For example, he quotes an interview with a worker from one of the research labs who said he vaccinated locals in Butare with Wistar’s vaccine in 1957. Hooper corroborates this with interviews of community members in eight villages around Butare, finding “two old men [who] independently told us that they recalled oral vaccinations against *mbasa*, or polio.” These data are then linked to the epidemic: In 1984, 88%

of prostitutes in Butare were HIV positive, “an extraordinary percentage for so early in the AIDS epidemic” (Hooper 2001a, 806).

7. The Royal Society meeting has received a bread-and-butter STS work-up by Brian Martin (2001b).

8. Weiss wrote in a personal email to me that: “In 2001, I jumped off the fence on the polio vaccine hypothesis in favour of ‘disproved.’ . . . But I am open to persuasion that my conclusion was premature” [email, 11/12/2017]. In his published article on the vaccine sample testing, he writes that the new data “may not convince the hardened conspiracy theorist,” and in a much cited passage, Weiss concludes that “some beautiful facts have destroyed an ugly theory” (Weiss, 2001b, 1036). Hooper’s “towering achievement” had become an “ugly theory” in Weiss’s estimation. Also worthy of note is Weiss’s comment that: “I would not have trusted Koprowski more than I could throw him” [interview, 12/13/2017].

9. Interview, 12/13/2017.

10. Interview, 12/13/2017.

11. Cutter Labs resorted to some creative legal reasoning to argue that they should not be liable. At the time, blood—typically human blood and plasma products used in medical treatments—was shielded from the implied warranties usual for consumer products. Cutter Labs tried to make the case that they should not pay damages for a child’s paralysis resulting from the vaccine since the manufacturing process used a horse serum—a blood component—and thus should fall under the blood shield (*Gottsdanker v. Cutter Laboratories* 1960).

12. There is now more literature and debate about where the SIV infected chimps may have originated.

13. The monkeys imported from India were gang caged, thus enabling the SV40 virus to spread among them. Vaccine companies used different techniques to make the vaccines. Vaccines made with one kidney had a 20% contamination rate, with two to three animals a 70% contamination rate, and when 10-plus animals were used, a 100% contamination rate. “Studies estimate that the vaccine infected between 10–30 million adults” (in itself a tellingly vague estimate), and that “potentially contaminated vaccine had been administered to almost 90% of individuals under 20” (Shah and Nathanson 1976, 5).

14. SV40 is often mentioned in the literature critical of vaccines. In my view, this is a result of virology and vaccinology having represented itself and its history as one of unalloyed success, which, even to the casual observer, is simply untrue. This gap can lead to overblown anxieties in the public domain. Formally acknowledging accidents and risks, and then showing how regulations have changed, may offer a better strategy against anti-vaxxers than flat-out denial.

15. Some 25 years after their initial use, an SIV *was* found in African Green monkeys (Weiss 2001b, 1035).

16. If the legacy of such fundamental confusions plagues the history of vaccinology, so, too, do they haunt the history of HIV/AIDS more generally. Consider, for example, the confusion over the identification of the virus in the 1980s and Robert Gallo’s attempts to take credit for being the first to isolate the virus over Luc Montanier (Crewdson 2003). From another angle, the myth of “Patient Zero,” the French Canadian flight attendant misrepresented as spreading hundreds of cases of AIDS to gay men in North America, had a long life that hasn’t entirely fizzled out

despite overwhelming evidence that this character was actually a fantasy made up by a journalist and his marketing department to sell books (McKay 2017). These examples illustrate the multiple stakes, not always benevolent, in origin stories.

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